My Child Has...

Intestinal Atresia and Stenosis

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What are intestinal atresia and stenosis?

Intestinal atresia is a congenital (present at birth) malformation that involves the complete absence or closure of a part of the intestine. Intestinal stenosis is a narrowing of the intestine that causes partial blockage. Intestinal atresia is common in the duodenum, jejunum and ileum, which make up the small intestine and is rare in the colon. Intestinal atresia accounts for 33 percent of all cases of intestinal obstruction at birth.

If not treated promptly, intestinal atresia and stenosis can lead to damage of the intestines.

How often does intestinal atresia and stenosis occur?

Intestinal obstruction occurs in approximately 1 out of every 1500 live births.

What causes intestinal atresia and stenosis?

The cause of most types of intestinal atresia is secondary to an in utero vacular accident. Obstruction of the jejunum may occur in response to another congenital anomaly, such as volvulus (a twisting of the intestine), malrotation of the intestine, hernia, intussusception, or abdominal wall defects such as gastroschisis or omphalocele, that can involve strangulation of the small intestine, which interrupts its blood supply.

About half of infants born with duodenal obstruction are premature, more than 30 percent have Down's syndrome and 50 to 75 percent have associated anomalies including cardiac, renal, and other gastrointestinal defects.

What are the symptoms of intestinal atresia and stenosis?

The symptoms of intestinal atresia and stenosis depend on the cause, where in the intestine the obstruction is and how long the condition is left untreated. The classic symptoms include:

- Failure to tolerate feedings
- Nausea and Vomiting
- Bilious vomiting
- Intermittent, abdominal pain
- Abdominal distention (sometimes)

Symptoms of intestinal atresia and stenosis may resemble other conditions or medical problems.

Please consult your child's physician for a diagnosis.

How is intestinal atresia and stenosis diagnosed?

With increasing frequency, these kinds of obstructions are identified on prenatal ultrasound after the finding of polyhydramnios (excess amniotic fluid), which is caused by the intestine's failure to properly absorb amniotic fluid. If your doctor suspects intestinal atresia or stenosis, your newborn will undergo the following diagnostic procedures after he/she is stabilized:

- Abdominal X-ray:
- lower GI (gastrointestinal) series (also called barium enema) A procedure that examines the rectum, the large intestine, and the lower part of the small intestine. A fluid called barium (a metallic, chemical, chalky, liquid used to coat the inside of organs so that they will show up on an x-ray) is given into the rectum as an enema. An x-ray of the abdomen shows strictures (narrowed areas), obstructions (blockages), and other problems.
- Abdominal ultrasound - A diagnostic imaging technique which uses high-frequency sound waves and a computer to create images of blood vessels, tissues, and organs. Ultrasounds are used to view internal organs as they function, and to assess blood flow through various vessels. Gel is applied to the area of the body being studied, such as the abdomen, and a wand called a transducer is placed on the skin. The transducer sends sound waves into the body that bounce off organs and return to the ultrasound machine, producing an image on the monitor. A picture or video tape of the test is also made so it can be reviewed in the future.

Other imaging studies of the cardiac and renal regions may also performed after the baby is stabilized to check for anomalies associated with intestinal atresia. Approximately one-third of the infants with intestinal atresia will have an associated life-threatening anomaly.

How is intestinal atresia and stenosis treated?

Specific treatment for intestinal atresia will be determined by your child's physician based on the following:

- the extent of the problem
- location of the obstruction
- your child's age, overall health, and medical history
- the opinion of the physicians involved in the child's care
- your opinion or preference

Duodenal atresia and stenosis

The initial treatment for duodenal atresia or stenosis involves stabilizing the baby. This process involves the decompression of excess gas. A baby with this condition has a distended and gas-filled stomach. The gas is decompressed through a tube that is placed into the stomach through the nose or mouth. Intravenous fluid replacement is also administered to replace vital electrolytes that a baby with this condition typically loses due to vomiting. Once the baby is stabilized, surgery is performed to fix the obstruction. Surgery involves excision of the obstructed part of the duodenum, followed by rejoining or reconnection of the healthy parts of the duodenum. In addition, a tube may be temporarily implanted through a surgical opening in the abdominal wall (gastrostomy), to drain the stomach and protect the airway. Nutrition will be administered intravenously until the infant is able to feed orally.

Jejunal and Ileal Atresia and Stenosis

Babies with this kind of obstruction must also be initially stabilized before the problem is corrected. This usually involves intravenous fluid replacement to maintain adequate fluid and electrolyte balance. Antibiotics will also be administered to treat any infections that result from this condition. Surgical correction involves the resection of the dilated proximal portion of the bowel followed by rejoining of the two ends of the bowel.

Nutrition

In children who have had an intestinal atresia or stenosis surgically corrected, feedings are started when a bowel movement is observed and gastric drainage and volume are minimal. Your baby's doctor will start your baby on a special formula that can be easily digested. When increases are tolerated, the density and volume can be increased to full strength.

What is the long-term outlook for a child with this condition?

How these babies fare heavily depends on whether there is an associated anomaly and whether or not the baby is left with an adequate length of intestine. Most babies do well, but some complications can occur such as gastroesophageal reflux. Your child's doctor will want to monitor your baby regularly after surgery.

Alimentary tract and pancreas
Alimentarni trakt i pankreas
ARCH GASTROENTEROHEPATOL 2002; 21 (No 1 – 2):

Stenosis of the small intestine and entero-enteral fistula due to mesenteric vascular occlusion

Stenoza tankog creva i entero-enteralna fistula prouzrokovani mezentericnom vaskularnom okluzijom

(accepted April 24th, 2002)

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Entero-enteral fistula due to mesenteric occulsion

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Gastroenterloska sekcija SLD-01728, 2002.

ABSTRACT

Stenosis of the small bowel due to mesenteric ischemia is rare and, to our best knowledge, a case of the entero-enteral fistula caused by that condition has not been described in literature so far. According to the clinical picture, patological features and localization, small bowel ischemic strictures can be divided into two groups: First, where ischemia goes unnoticed with symptoms only when intestinal stenosis is developed and where a short segment of the bowel is affected, and second, with episodes of acute ischemia followed by asymptomatic periods as well as by symptoms of intestinal obstruction, where the involved segment is longer and usually localized in jejunum. The diagnosis is based on the history, enteroclysis and pathohystological examination. In the differential diagnosis Crohn's disease and others causes of the acquired stenosis of the small bowel have to be considered. The therapy is surgical implying the removal of the stenosed segment of the small bowel with the additional angioplastical operation, when

necessary.

We present a case of 66-year old patient previously treated for mesenteric vascular occlusion with instillation of Novocain solution in the radix of the mesentery. The diagnosis of intestinal stenosis was established by upper GI series and confirmed itraoperatively. Double jejunal stenosis was found. Segmental resection of the affected intestine and end-to-end anastomosis were performed. Examination of the resected specimen revealed a jejuno-jejunal fistula in the stenotic segments. Crohn's disease was ruled out. The postoperative course was uneventful and the patient was discharged several days later. He is symptom-free three months after surgery.

Key words: mesenteric ischemia, intestinal obstruction, entero-enteral fistula.

SAZETAK

Stenoze tankog creva prouzrokovane mezenterijskom sudovnom ishemijom su retke. Prema nasem uvidu u medicinsku literaturu slucaj entero-enteralne fistule prouzrokovane ovakvim stanjem do sada nije opisan. Zavisno od klinicke slike, patoloskih promena i lokalizacije, ishemijske strikture tankog creva mogu da se podele u dve grupe. U prvu grupu spadaju ishemije koje prolaze klinicki nezapazeno i koje se ispoljavaju tek onda kada nastanu simptomi stenoze tankog creva. U drugoj grupi su slucajevi u kojih su epizode crevne ishemije manifestne i iza kojih sledi asimptomski period sve do ispoljavanja simptoma i znakova stenoze tankog creva. Dijagnoza se zasniva na anamneznim podacima, enteroklizi, i patohistoloskom pregledu reseciranog dela creva. U diferencijalnoj dijagnozi uvek treba da se razmotri Crohn-ova bolest i drugi uzroci stecenih stenoza tankog creva. Lecenje je hirursko i podrazumeva odstranjenje stenozantnog segmenta sa dodatnom angioplastikom ukoliko je to indicirano.

U radu se prikazuje slucaj pacijenta zivotne dobi 66 godina koji je prethodno bio lecen od mezenterijske vaskularne okluzije instilacijom Novocaina u radiks mezenterijuma. Dijagnoza intestinalne stenoze je postavljena radioloskim pregledom alimentarnog trakta. Itraoperativno je otkrivena dvostruka stenoza jejunuma. Segmentna resekcija izmenjenog dela creva i termino-terminalna anastomoza je bila nacinjena. Pregled reseciranih delova creva je otkrio jejuno-jejunalnu fistulu u stenoticnom segmentu. Crohno-va bolest je bila iskljucena. Postoperativni tok je bio bez komplikacija i pacijent je otpusten desetog dana izlecen.

Kljucne reci: mesentrijska ishemija, intstinalna opstrukcija, entero-enteralna fistula.

Ischemic disease of the small intestine is not frequent. In the acute form it is characterized with high mortality (over 50%). To our best knowledge, small bowel stenosis due to mesenterial ischemia is rare, while entero-enteral fistula caused by ischemia has not been described until now.

CASE REPORT

Four months before admission to our institution, the patient, a 66 years old man, undergone emergency explorative laparatomy due to simptoms and signs of mesenteric ischemia. The patient has passed medical history of arterial hypertension for about 30 years. He experienced acute myocardial infarction in 1979, and in 1983 a triple arteriocoronary bypass due to coronary disease. He never used digitalis or potassium chloride medication. At operation mesentery radix was infiltrated with Novocain solution resulting in improvement of bowel perfusion thus making the bowel resection unnecessary. Three months later he became pyrexial. Nausea and protracted diarrhoea appeared as well.

During the following month, most of the symptoms abated except abdominal colic, malaise, and progressive weight loss. Physical examination revealed no abnormalities except mild tenderness of the lower abdomen. Laboratory findings were within the normal limits. Plain abdominal x-ray films demonstrated air-fluid levels. Barium enema and sygmoidoscopy were suggestive of dolichocolon. Examination of the small intestine with enteroclysis showed presence of two jejunal stenosis with large prestenotic dilatations. FIGURE 1. Doppler ultrasonography of portal system did not reveal any pathological findings. There were no signs of thrombosis, decreased flow or presence of collateral venous vessels. Abdominal aortography and selective visceral angiography showed: stenosis of the coeliac trunc up to 60-70%, occlusion of superior mesenteric artery in the middle part with rich collateral vascularisation and an arcus of Riolan that vascularised the coecum with normal features of mesenteric and portal veins. FIGURE 2. Accordingly, mesenteric ischemia was recognized as the aetiological factor. The patient was operated on 26.07.1999. After the adhesions were removed a subtotal occlusion of the small intestine, 80-100 cm of Treitz and an intestinal jejuno-jejunal fistula was found. FIGURES 3,4. Resection of the affected segment of the small intestine was performed in the length of 70 cm with a stapled side-to-side anastomosis. Postoperative recovery was uneventful. The patient was discharged 9 days after the operation and was symptom-free at the time. On the last follow-up examination the patient had no complaints. Histopathological examination of the resected part of the small intestine showed chronic inflammation of the mucosa with ulceration, Crohn's disease-like chronic lymphocytic inflammation of the submucosa without granulomata or other histopathological features of Crohn's disease. This was nonspecific finding that might correspond to the ischemic origin of the changes.

Figure 1. *Enteroclysis showing two jejunal stenosis with prestenotic dilatations*

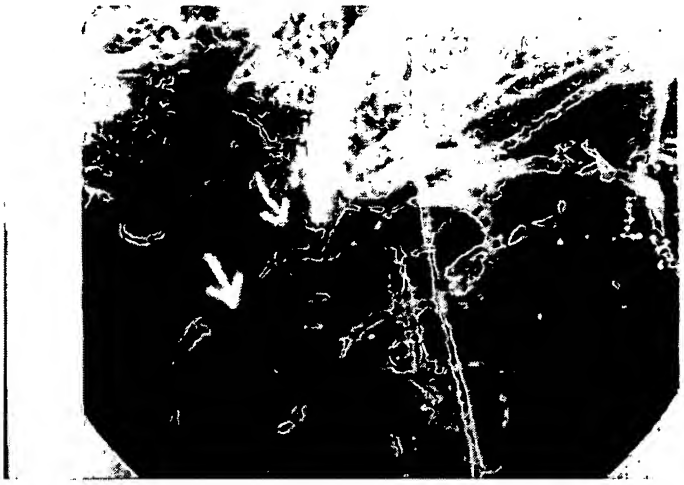

Figure 2. *Abdominal aortography and selective visceral angiography showing stenosis of the celiac truncus, occlusion of superior mesenteric artery with rich collateral vascularisation.*

Figure 3. *Stenosed jejunal segment with large prestenotic dilatation*

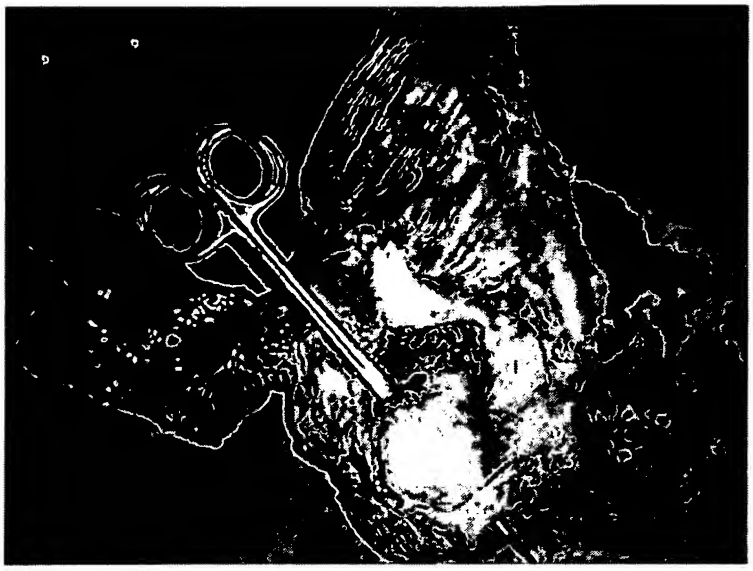

Figure 4. *Resected specimen showing stenosis of the small bowel with prestenotic dilatation and jejuno-jejunal communication (fistula).*

DISCUSSION

Mesenteric ischemia can be **chronic**, with functional bowel changes (malabsorption), but without impaired intestinal vitality or **acute**, where ischemic damage of the bowel is so severe that jeopardizes the vitality of the intestine thus leading to structural changes. In the acute mesenteric ischemia, ischemic damage may vary from transient impairment of the bowel function to transmural ischemic necrosis resulting in intestinal perforation. Between these two extremes is the so-called "subnecrotizing ischemia", when ischemic lesion does not damage all layers, but the mucosa only, which is the most vulnerable to ischemia (1). Resistance to ischemic damage rises from the lumen inside out. The level of ischemic damage progression depends on the degree of ischemia and of efficacy of compensatory mechanisms. When ischemic damage reaches the lamina muscularis mucosis, then it leads to formation of fibrous tissue and, subsequently, to retraction and formation of the bowel stenosis (2). Acute mesenteric ischemia usually is so severe that in more than 50% cases of cases it ends lethally (3). All causes of acute mesenteric ischemia belong to this type of ischemic damage. Thrombosis of the superior mesenteric vein is very rare (4). Embolisation of the superior mesenteric artery (SMA), thrombosis of the SMA, nonocclusive ischemic disease and, especially, focal segmental ischemia are causes of ischemic intestinal strictures (5,6,7). In embolisation of the SMA major emboli may be present in 85-90%, usually lodged in the beginning of ileocolic artery and minor emboli, present in 10-15%, in the distal part. The latter are mostly the cause of intestinal stenosis (2). Experimental studies support that opinion. Embolisation of branches of the SMA in dogs with gelatin sponges provoked segmental intestinal stenosis (8). Reasons for focal segmental ischemia of bowel include atheroembolism, strangulated hernias, vasculitis, blunt abdominal trauma, segmental venous thrombosis, radiotherapy, drugs (oral contraceptives, digoxins, NSAID, cocaine) where, due to a range of damage of the intestinal vascular bed, adequate collateral circulation prevents transmural necrosis of small intestine (2,9.10). That is the reason why acute focal segmental ischemia is probably the most common cause of the ischemic bowel stenosis. Clinical manifestations of mesenteric ischemia correlate with the level of ischemic damage of the bowel, and less with the underlying cause (2).

Based on histopathological findings, clinical picture and evolution of the disease, we can divide all acquired ischemic strictures of the small bowel into two major types (11):

1) Asymptomatic form of mesenteric ischemia. This clinically manifests with insidously but progressively developing intestinal obstruction. Usually short segment of the small intestine is affected (usually middle or terminal ileum). .
2) Symptomatic form is when symptoms and signs of the acute mesenterial ischemia are present from the beginning. This was followed by asymptomatic period and gradually developing symptoms and signs of the intestinal occlusion thereafter. In this case larger part of the bowel, oftenly jejunum, is affected.

It is not always possible to make a clear distinction between these two types of intestinal ischemic strictures, since transitive forms and variations are not rare (5,7,12,13). Our patient, however, fits into to this classification. Kradijan and associates described 6 patients with large ischemic lesions of the small intestine who had an asymptomatic phase followed by clinical picture of ileus and perforation in two cases. The strictures were localized in the ileum and involved short segments up to 3 cm in length, with normal appearance of bowel under stenosis. The confusion is produced by the fact that 5 of 6 patients had enteric-coated potassium chloride in therapy, later found to be the culprit of formation of strictures and bowel perforation, while the lesions completely matched the lesions accompanying this form of medication, in their appearance (pylorus-like annular and tubular stenoses) and localization (distal ileum) (14).

It difficult to determine why ischemic stricturing is more frequent in certain portions of the small intestine. This is partly due to poor vascularisation of the terminal part of the ileum and rich vascularization of the jejunum. That is the reason why larger segment of the jejunum can be involved with ischemic process without causing a perforation, but only stenosis. Macroscopic and radiographic appearance of this type, where stenosis involves long segments of the small bowel, is similar to findings in the Crohn's disease. Therefore, histopathological examination is necessary for differential diagnosis (1,5,11).

It is important to distinguish ischemic strictures from nonischemic ones due to Crohn's disease, neoplasm, other inflammatory tumors (for example, acute pancreatitis), use of enteric-coated potassium chloride, and cause of intestinal ischemia: trauma, irradiation, mechanical occlusion, vasculitis, drugs (NSAID's, digitalis, oral contraceptives), embolisation or thrombosis of the mesenteric artery, because further treatment depends on this (7,15).

The diagnosis of the intestinal stenosis is simple. Contrast radiography of the small bowel is the diagnostic modality of choice in such a case (16). Ischemic small intestinal lesions may often be the cause of the acquired small bowel stenosis and therefore angiographic examinations are necessary to diagnosed it.

The treatment includes resection of strictured segment of the small intestine and end-to-end anastomosis. Also, depending on the cause of ischemia, reconstructive surgery of mesenteric arteries directed towards improvement of perfusion of mesenteric vascular bed is required, as well discontinuation of related drugs. Therefore, depending of the cause of intestinal stenosis the type of treatment is not limited only to resection of stenotic segment.

In conclusion, our case of double jejunal stenosis and jejuno-jejunal fistula was caused by mesenteric ischemia, namely SMA thrombosis, which was of limited range due to the localization and previous intraoperative Novocain treatmen. According to these facts and to angiography findings, which showed well developed collateral circulation, the treatment was limited to resection of the stenotic segments. The continuity of digestive tract was established with termino-terminal jejuno-jejunal anastomosis.

REFERENCES:

1. Wayt DM, Helwig EB: Small-bowel ulceration- iatrogenic or multifactorial origin? Amer J Clin Path 1968,49:26-40.
2. Brandt LJ, Smithline AE: Ischemic lesions of the bowel. In Feldman M, Sleisenger MH, Scharschmidt BF (eds): Sleisenger and Fordtran's Gastrointestinal and Liver Disease: Pathophysiology, Diagnosis, Management, ed 6. Philadelphia, WB Saunders, 1998, pp 2009-2024.
3. Bastidas JA, Reilly PM, Bulkley GB: Mesenteric vascular insufficiency. In Yamada Y, Alpers DH, Owyang C et al (eds): Texbook of Gastroenterology, ed 2. Philadelphia, JB Lippincott, 1995, pp 2490-2523.
4. Eugene C, Valla D, Wesenfelder L et al. Small intestine stricture complicating superior mesenteric vein thrombosis. A study of three cases. Gut 1995; 37: 292-5.
5. Feurle GE, Haag B. Acute small bowel ischemia without transmural infarction. Z Gastroenterol 1991; 29: 349-52.
6. Thaker P, Weingarten L, Friedman IH. Stenosis of the small intestine due to nonocclusive ischemic disease. Arch Surg 1977; 112: 1216-7.
7. Saegesser F, Borgeaud J, Schnyder P et al. Stenosis of the small intestine of ischemic origin in the adult. Schweiz Med Wochenschr 1976; 106: 367-76.
8. Cho KJ, Schmidt RW, Lenz J. Effects of experimental embolization of superior mesenteric artery branch on the intestine. Invest Radiol 1979; 14: 207-12.
9. Allen JC. Post-traumatic small bowel obstruction. J R Army Med Corps 1994; 140: 47-8.
10. De Backer AI, De Schepper AM, Vaneerdeweg W, Pelckmans P. Intestinal stenosis from mesenteric injury after blunt abdominal trauma. Eur Radiol 1999; 9: 1429-31.
11. Mozes M, Adar R, Tsur N et al. Intestinal obstruction due to mesenteric vascular occlusion. Surg Gynecol Obstet 1971; 133: 583-7.
12. Haraguchi M, Matsushima S, Fujie Y, Sugimachi K: Ischemic stricture of the jejunum-report of a case. Jpn J Surg 1990,20:715-719.
13. Kradjian RM. Ischemic stenosis of small intestine. Arch Surg 1965; 91: 829-34.
14. Grosdidier J, Boissel P, Bresler L, Vidrequin A. Stenosing and perforated ulcers of the small intestine related to potassium chloride in enteric-coated tablets. Apropos of 11 cases. Chirurgie 1989; 115: 163-9.
15. Kato T, Morita T, Fujita M et al. Ischemic stricture of the small intestine associated with acute pancreatitis. Int J Pancreatol 1998; 24: 237-42.
16. Ginai AZ, Hussain SM, Hordijk ML, den Hollander JC. Case report: solitary ischaemic small bowel stenosis. Br J Radiol 1994; 67: 405-7.
17. Lietz H, Meissner K. Mysterious segmental stenosis of the small intestine. Dtsch Med Wochenschr 1982; 107: 299-303.

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Figure 3. Stenosed jejunal segment with large prestenotic dilatation.

Figure 5. Resected specimen showing stenosis of the small bowel with prestenotic dilatation and jejuno-jejunal communication (fistula).

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Display Abstract Show

Intestinal stenosis and perforating complications in Crohn's disease.

Makowiec F, Jehle EC, Koveker G, Becker HD, Starlinger M.

Department of General Surgery, University of Tubingen, Germany.

The charts of 384 patients with Crohn's disease were reviewed to assess the prognostic value of a bowel stenosis documented at the time of initial diagnosis for the occurrence of perforating (abscess, fistula, free perforation) or obstructing complications requiring surgical intervention. Mean follow-up was 5.6 years. At time of diagnosis a bowel stenosis (S) was documented in 143 patients (37.2%). 130 patients underwent surgery, 62 (48%) for obstruction, 18 (14%) for a perforating complication, 12 (9%) for both obstructing and perforating complication and 38 (29%) for intractable disease. The cumulative rates of surgery were calculated using lifetable analysis. The presence of a stenosis at the time of initial diagnosis was a risk factor for the likelihood of surgery overall [65% (S) vs. 40% (no S) after 10 years; $P < 0.001$] and of surgery for obstruction [70% (S) vs. 34% (no S); $P < 0.001$] but did not increase the likelihood of a perforating complication [24% (S) vs. 29% (no S); n.s.]. A perforating complication requiring surgery may therefore not be predicted by the mere diagnosis of a stenosis. Prophylactic surgery of stenotic lesions in patients with Crohn's disease to prevent the development of a perforating complication therefore is not recommended.

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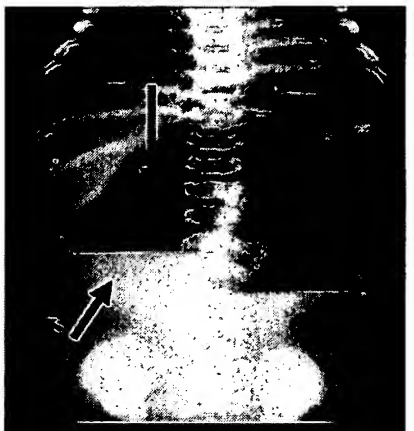
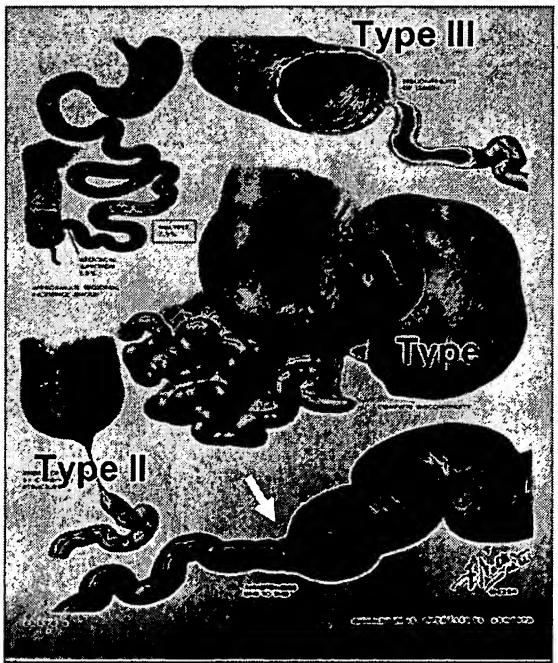
Intestinal Atresia or Stenosis

Occasionally, the diagnosis of jejunoileal atresia (blockage of the small intestine) may be suspected when polyhydramnios (excess fluid around the baby and enlarged loops of intestine are observed on ultrasound on the fetus. Typically, however, a newborn with jejunoileal atresia presents with green/yellow vomiting, distention of the abdomen, and failure to have a bowel movement on the first day of life. Distention of the abdomen may vary depending on the level of the blockage. Although atypical, passage of stool may occur in the setting of a jejunoileal atresia which has developed later in gestation. X-rays of the abdomen often demonstrate a single large loop of enlarged, air-filled intestine just upstream from the atresia. An X-ray with dye placed into the rectum eliminates the possibility of blockage of the large intestine and typically an unusually small colon unless the blockage occurred late in gestation.

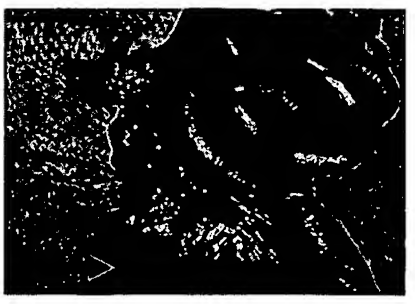


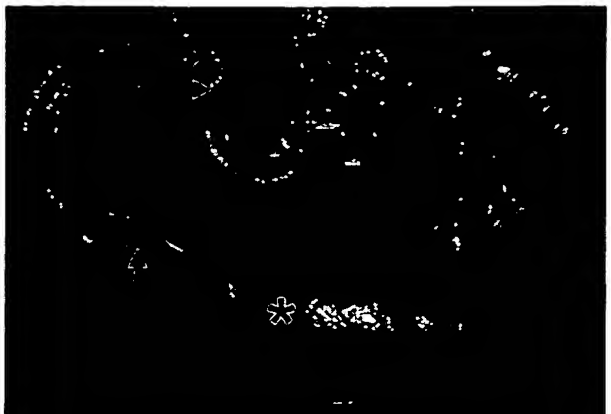
X-ray of a newborn with jejunoileal atresia. Note the huge portion of the intestines with air (red arrow) and fluid (blue arrow).



A Netter depiction of jejunoileal atresia. The three types (I, II, III) are demonstrated as is the intestines after the blockage is removed and the ends of the intestine joined (arrow).

Three different types of jejunoileal atresia are observed: Type I (19%) consists of a single, thin web or diaphragm which blocks the intestine; type II (31%) a cord of scar between the upstream and downstream ends; and type III (50%) is an atresia in which a portion of the intestine is completely missing. All are treated similarly. A timely, but not emergent, operation is typically performed. An incision with upper abdomen typically reveals a very large upstream bowel and a small downstream portion of the intestine. Some of the upstream portion must be removed because it will not work correctly. The two ends will still be different in size and, therefore, a special "end-to-back" joining of the two ends must be performed. Since between 6-20% of newborns may have more than one atresia, downstream small intestine must be insufflated with water or air and examined carefully for the presence of another blocked area of intestine. Blockage of the colon is rarely observed, but the treatment is similar to that of jejunoileal atresia.





Note the huge portion of the intestine (*) and the multiple jejunoileal atresias (arrows).

Survival or complications are generally affected by associated problems such as prematurity, heart disease, or insufficient intestine (short gut syndrome). Problems where the intestinal ends are joined, such as leak or stricture (narrowing), occur in approximately 5% of cases. It commonly will require a number of days to weeks before feeding may be tolerated. Most infants with more than 15 cm of remaining small intestine and an ileocecal valve (where the small intestine meets the large intestine) or more than 40 cm of small intestine if the ileocecal valve has been removed will survive.

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